Welcome to Q Lab

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Research interests:
Research in Dr. Yang’s lab focuses on exploring molecular mechanisms underlying the development and progression of heart failure, especially those related to transcriptional regulation of myocardial fatty acid and carbohydrate metabolisms (e.g., PPAR signaling pathway) in pathological conditions such as hypertension, obesity and diabetes. His lab uses cutting-edge technologies in molecular genetics to generate and study transgenic and knockout mouse models. A variety of techniques in molecular and cellular biology, biochemistry and Pathophysiology are used to assess these mouse models in an effort to identify mechanisms and get insights into novel therapeutic strategies.

Current Projects
• PPAR signaling in regulation of myocardial lipid and energy homeostasis in pathophysiological conditions (supported by R01 HL085499 and R01 HL084456)
• Effects of Salacia oblonga root extract on cardiac hypertrophy through regulating myocardial metabolisms (supported by R21 AT003734)

Laboratory Techniques:
There are four areas of technical strengths in our laboratory: 1) molecular biology including most of the routine DNA, RNA and protein techniques; 2) development and characterization of animal models through conventional and conditional transgenic and gene targeting approaches; 3) Biochemical assays assessing enzyme activities, Glucose tolerance assays and hyperinsulinemic euglycemic clamp techniques, mitochondrial function and substrate utilization in isolated working heart and cultured adult cardiomyocytes; 4) Mouse cardiac physiology analyses in cell, organ and whole animal levels; 5) Small animal surgeries: TAC, LAD ligation, ovariectomy, etc.

Recent transgenic lines created by the Qlab: VP16-PPARδ (VPD), VP16-PPARγ (VPG), VP16-LXRα (VLA), VP16-LXRβ (VLB)

Methodology for the above transgenic lines, use VPG line as example:
An inducible, cardiomyocyte-restricted transgenic line with hyper-expression of a constitutively active PPARγ

We generated the TG mouse model with tamoxifen inducible cardiomyocyte-restricted overexpression of a constitutively active mutant PPARγ gene (VP16-PPARγ), which is created by fusing the activation domain of the herpes simplex virus Vp16 protein to the N-terminal of mouse PPARγ. This mutant (VP16-PPARγ, designated as VPG) can transactivate a PPARγ reporter to a similar magnitude as to what is observed with the natural receptor in the presence of ligand (Saez et al., 2004). More importantly, VP16-PPARγ only signals through PPARγ pathways. The transgenic construct of VPG mice is shown. Before recombination, the loxP-flanked CAT gene is expressed under the control of the The CMV early enhancer/chicken β actin (CAG) promoter, which drives ubiquitous transgenic expression. The transgene is silent without the removal of the CAT gene. Cre-mediated recombination results in the deletion of the CAT gene and the expression the VP16-PPARγ genes. Overexpression of this mutant form of PPARγ should ensure the constitutive transactivation of its target genes. For cardiomyocyte-restricted hyper-expression of the VP16-PPARγ, the VPG mice were then crossed with the αMyHC-Mer-Cre-Mer (MCM) transgenic mice (Sohal et al., 2001). Genomic DNA isolated from mouse tails was subjected to PCR analysis to get MVPG mice. Tamoxifen (Sigma) (80µg/g of body weight/day) was administered by intraperitoneal injection once daily for 5 days to induce cardiomyocyte-restricted PPARγ overexpression in adult mice.

**Current Lab members:**
Lab manager:
Olga Zhelyabovska, Ph.D.

Research Assistants:
Huan Yang
LaShonda Silmon

Graduate students:
Jinwen Luo
Sijie Wu

Post-doctoral fellows and Research Associates:
Peiyong Wang, MD, PhD
Jian Liu, MD, PhD
Lan He, MD, PhD
Yiqun Zhou, PhD
Teayoun Kim, PhD
**Selected Publications:**


Li Y, Cheng LH, Qin QH, Liu J, Lo WK, Brako LA and Yang Q: High fat feeding in cardiomyocyte-restricted PPARδ knockout mice leads to cardiac overexpression of lipid metabolic genes but fails to recue cardiac phenotypes. *Journal of Molecular and Cellular Cardiology* 2009:47(4):536-43

**Research Positions Available**

Postdoctoral positions are available in this lab. Candidates should possess a recent PhD or MD/PhD and have a strong research background in molecular biology and/or experience in cell biology, biochemistry and cardiovascular pathophysiology.
Please send curriculum vitae and names of three references, preferably by e-mail or FAX, to: Dr. Q Yang, Department of Nutrition Sciences, The University of Alabama at Birmingham, 1675 University Blvd, Webb 435, Birmingham, AL 35294-3360
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